Atty Dkt. No.: UCAL-234

USSN: 09/884,875

## **AMENDMENTS**

## IN THE CLAIMS

1. (Currently Amended) A method for identifying an agent that modulates NF-κB activity in transcription of a gene in a eukaryotic cell, the method comprising:

contacting a candidate agent with a eukaryotic cell in vitro, the eukaryotic cell having with acetylated RelA, deacetylated RelA, or both acetylated and deacetylated RelA; and detecting a level of deacetylated RelA;

wherein detection of an increase <u>in the level</u> of deacetylated RelA in the presence of the candidate agent compared to a level of deacetylated RelA in the absence of the candidate agent indicates that the agent inhibits activity of NF-kB in gene transcription <u>in the eukaryotic cell</u>.

- 2. (Original) The method of claim 1, wherein RelA is detectably labeled so that deacetylation results in release of the detectable label from RelA, and said detecting of deacetylated RelA is by detecting a decrease in detectably labeled RelA.
- 3. (Original) The method of claim 1, wherein RelA is detectably labeled so that deacetylation results in release of the detectable label from RelA, and said detecting of deacetylated RelA is by detecting released detectable label.
- 4. (Currently Amended) The method of claim 1, wherein said detecting <u>is</u>

  <u>performed of deacetylated RelA is compared to a level of deacetylated RelA</u> in the presence of histone deacetylase 3 (HDAC3).
- 5. (Currently Amended) The method of claim 1, wherein RelA is within a eukaryotic cell and detecting detection of deacetylated RelA is by detection of export of RelA from the nucleus of the cell, wherein detection of RelA export indicates RelA is deacetylated.
- 6. (Currently Amended) The method of claim 1, wherein RelA is within a eukaryotic cell and detecting detection of deacetylated RelA is by detection of an increase in RelA binding to IkBa.

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7. (Currently Amended) A method for identifying a substance that inhibits NF-kB activity, comprising testing a substance for activity in deacetylation of RelA or inhibition of RelA acetylation, the method comprising the steps of:

exposing a sample comprising RelA to a test substance;

comparing the level of deacetylated RelA in the sample comprising the test substance to the level of deacetylated acetylation of RelA in a sample without the test substance; and

determining whether the test substance provides for a level of deacetylated RelA is greater in the sample exposed to the test substance than a level of deacetylated RelA in the sample without absence of the test substance;

wherein <u>an increase in activity of the test substance in increasing</u> deacetylated RelA <u>in</u> <u>the presence of the test substance</u> indicates the test substance inhibits NF-κB activity.

- 8. (Original) The method according to claim 7, wherein the exposing step includes using an extract of cells, which were treated with an inducer for NF-kB activation, or a fraction of said extract.
- 9. (Original) The method according to claim 7, wherein a cell-free system is used for the exposing step.
  - 10. (Original) The method according to claim 9, wherein RelA is bound to a support.
  - 11-18 (Cancelled)
- 19. (Original) The method of claim 1, wherein said contacting is in the presence of a protein or protein complex that acetylates RelA.
- 20. (Previously Presented) The method of claim 19, wherein the protein that acetylates RelA is CBP or p300.
- 21. (Previously Presented) The method of claim 1, wherein RelA is within a eukaryotic cell, which cell contains CBP and p300.

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- 22. (Currently Amended) The method of claim 1, wherein said contacting is in the presence of HDAC3 and wherein detection of an increase of deacetylated RelA in the presence of the candidate agent and <u>HDAC3 HDAC2</u> is compared to a level of deacetylated RelA in the absence of the candidate agent and the presence of HDAC3.
- 23. (Previously Presented) The method of claim 8, wherein the extract comprises p300 and CBP.
- 24. (Previously Presented) The method of claim 23, wherein the extract comprises HDAC3.